

Torsade de pointes, an atypical ventricular tachycardia

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'What's in a name? That which we call a rose
By any other name would smell as sweet'

W. Shakespeare, *Romeo and Juliet*, II, ii, 43

When defining a 'new' arrhythmia it is reasonable to suppose that it may indeed have been recognized many years previously. This applies to the disorder now called *torsade de pointes*, whose features have been so well worked out since it was characterized by Dessertenne (1966, 1967). As the opening quotation implies, why introduce a new name? The specific characteristics of this arrhythmia are poorly recognized in English and American reports and the importance of principles relating to its aetiology and management are so crucial that it deserves to be better known; as its mechanism is not yet quite clear a morphological name seems more appropriate. Reference to a standard French-English dictionary (*Harrap's New Shorter French and English Dictionary*, 1967) shows how well *torsade de pointes* describes the appearances: '*torsade*, twisted fringe, . . . twist, coil, . . . thick bullion (of epaulet); *pointe*, point (of pin, sword, etc.), tip, head (of arrow, lance) . . .'. Its use in French is much more euphonious than translations like 'wave bursts'.

Though the electrocardiographic features were first noted more than 50 years ago (MacWilliam, 1923; Wiggers, 1929), it is only since Schwartz, Orloff, and Fox (1949) and Schwartz and Hallinger (1954) described 'transient ventricular fibrillation' that its importance has been recognized. Nevertheless, the syndrome still receives inadequate attention: it is not mentioned in a recent otherwise excellent editorial on ventricular tachycardia (Fisch and Noble, 1975), and examples are published under other guises in Anglo-American textbooks on

electrocardiography or arrhythmias. If, however, we turn to the French literature for authoritative reviews (Brochier, Motté, and Fauchier, 1972; Slama *et al.*, 1973; Puech, 1974), apparently obscure ventricular tachyarrhythmias that have proved difficult to classify become understandable (Bleifer *et al.*, 1974; Pilling and Nanton, 1974; Meijler, Robles de Medina, and Zimmerman, 1975; Schamroth, 1975).

Diagnostic features

The main features have been summarized and depicted diagrammatically elsewhere (Krikler, 1974). The diagnosis is made from the electrocardiographic appearances both during and between attacks and from any aetiological factors (Slama *et al.*, 1973). The specific findings (best seen when multiple simultaneous leads are taken) consist of paroxysms of ventricular tachycardia in which the QRS axis undulates over runs of 5 to 20 beats, with definite changes in direction (Fig.). This has led to the use of a variety of descriptive terms for what are clearly isolated examples of this disorder, including 'paroxysmal ventricular fibrillation' (Loeb *et al.*, 1968), 'transient recurrent ventricular fibrillation' (Tamura *et al.*, 1967) and 'cardiac ballet' (Smirk and Ng, 1969). But these morphological criteria can be deceptive, since *torsade de pointes* is sometimes associated with fairly long runs of uniform QRS complexes and conventional ventricular tachycardia can be bidirectional (Slama *et al.*, 1973).

Attacks may be brief, consisting of only a few complexes (when diagnosis may be difficult and be

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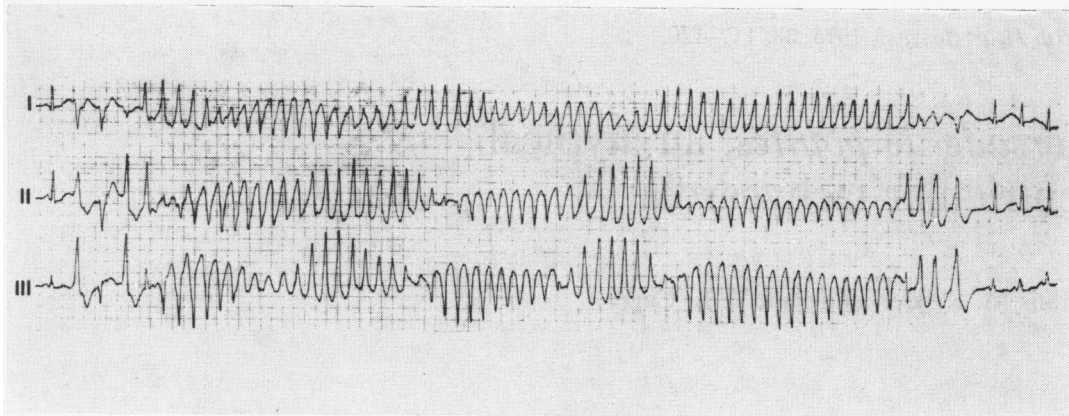


FIG. Simultaneous recording of leads I, II, and III from a patient with Conn's syndrome showing torsade de pointes. Sinus rhythm is shown in the first, and in the last three, beats in which the QT appears prolonged; precise measurement is impossible because the succeeding P waves are merged with the preceding T waves. The first four beats of the arrhythmia show tachycardia, bidirectional in leads II and III, succeeded by a run of tachycardia with wide QRS complexes in which the QRS axis rotates, in an undulating fashion, the differences being well shown by comparison of the three leads. The last run of torsade de pointes is prolonged, and the QRS complex is more uniform, with a closer resemblance to classical ventricular tachycardia, the arrhythmia terminating with another change in axis for the last four beats.

aided by identification of the underlying features), or prolonged, as in the Figure, when syncope may result. When an attack stops spontaneously it is often with a ventricular complex intermediate in form between the QRS of the basic tracing and that seen in tachycardia, though sometimes the final beat is identical to the initiating ventricular extrasystole. Some cases do, however, progress to ventricular fibrillation, and death is an ever-present risk in untreated *torsade de pointes*.

The start of the tachycardia is of both diagnostic and pathogenetic importance. Whereas ventricular tachycardia may follow an early ventricular extrasystole falling on the T wave of the preceding sinus beat (Smirk, 1949), in *torsade de pointes* the initiating extrasystole is usually late, and if, as is often the case, the QT interval is prolonged there is a longer period in which asynchrony of cardiac activity exists (Pick and Katz, 1963). It is tempting to describe the appearances as indicating chaotic cardiac action but, as Dessertenne (1973) points out, the features reflect a well-organized electrical process: clearly there is mechanical inefficiency leading to poor cardiac output, often with syncope. We have noted the initiation of *torsade de pointes* in four patients as a result of intracardiac electric stimulation (Evans *et al.*, 1976), and think that this supports deductions—that can be made from the morphological appearances and the fact that there is usually underlying evidence of impaired repolarization—

that the arrhythmia is due to a re-entry mechanism (Raynaud *et al.*, 1969).

Aetiology

A cardinal aspect of *torsade de pointes* is the underlying aetiological situation, which can often be seen or suspected in the basic electrocardiogram of the patient (Table). Of major importance are bradycardias, very often high-grade atrioventricular or sinoatrial block. While the precise mechanism is unclear, it has been suggested that this favours desynchronization of cardiac activation (Han and Moe, 1964; Han *et al.*, 1966; Motté *et al.*, 1970): the occurrence of ventricular tachycardia in a patient suffering from heart block suggests that it is *torsade de pointes*. Hypokalaemia may also provide electrocardiographic clues and should be excluded in all cases of *torsade de pointes*; retrospective analysis of hypokalaemia-induced ventricular arrhythmias may show them to have been caused by *torsade de pointes* (Tamura *et al.*, 1967), and we have recently found *torsade de pointes* caused by Conn's syndrome and familial periodic paralysis (Krikler *et al.*, 1976). While hypomagnesaemia is much less common—and certainly less often looked for—it has been identified as a cause of *torsade de pointes* (Loeb *et al.*, 1968).

Ventricular tachycardia is clearly a major cause of death in patients with the congenital syndromes in

TABLE Recognized causes of torsade de pointes

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- 1) Slow basic rhythm
 - a) Sinoatrial depression/disease
 - b) High-degree AV block
 - 2) Electrolyte deficit(s)
 - a) Potassium
 - b) Magnesium
 - 3) Congenital QT prolongation syndromes
 - a) Overt, with deafness
 - b) *Forme fruste*
 - c) Concealed
 - 4) Drugs
 - a) Cardioactive agents: quinidine, lignocaine, procainamide, prenylamine, etc.
 - b) Psychotropic agents: phenothiazines, *tricyclic antidepressants, ? other major tranquilizers
 - 5) Cardiac ischaemia
 - 6) Myocarditis
-

which the QT interval is prolonged, whether with deafness (Jervell and Lange-Nielsen, 1957) or without (Romano, Gemme, and Pongiglione, 1963; Ward, 1964); indeed it may be present as a *forme fruste*, with QT prolongation only apparent after exercise (Von Bernuth *et al.*, 1973). In at least some of these cases the ventricular arrhythmia appears to be *torsade de pointes* (Motté *et al.*, 1970).

For many years ventricular tachyarrhythmias have been known as a complication of quinidine therapy (Levy, 1922), and as this drug acts in part by prolonging repolarization it is not surprising that *torsade de pointes* should be seen (Acierno and Gubner, 1951; Rainier-Pope *et al.*, 1962), more often as a result of overdosage than idiosyncrasy (Brochier *et al.*, 1972). Though other drugs with an action resembling that of quinidine were not noted as causes of *torsade de pointes* by Brochier *et al.* (1972), we have seen it twice (a new observation, which makes it less than coincidence) after an overdose of lignocaine (unpublished observations). Several cases of the arrhythmia have been caused by prenylamine, possibly because of its quinidine-like effects (Bens *et al.*, 1973). In its own right digitalis has not been incriminated, and we would not expect it to cause *torsade de pointes* as it shortens the repolarization time. If it occurs attention should be focussed on any concomitant medications—for example, potassium-losing diuretics or quinidine. Among non-cardiac drugs that may induce *torsade de pointes* are phenothiazines, notably thioridazine (Schoonmaker, Osteen, and Greenfield, 1966), and tricyclic antidepressants. We have seen *torsade de pointes* followed by ventricular fibrillation after an overdose of amitriptyline (unpublished observations): in sinus rhythm the QT had been prolonged, and this persisted when haloperidol was

substituted, the interval returning to normal when all psychotropic agents were stopped.

Torsade de pointes is a known but rare complication of myocardial infarction (Dalle, Meltzer, and Kravitz, 1967), occurring so seldom perhaps because of lack of associated asynchrony of repolarization (Puech, 1974), but it has been recognized during the course of so-called Prinzmetal (variant) angina. When this is associated with paroxysmal atrioventricular block (Chiche, Haiat, and Steff, 1974) the pathogenesis could be that of atrioventricular block of any cause, but its occurrence in this disorder in the absence of heart block (Benaim *et al.*, 1975) implies the presence of subtle factors encouraging re-entry. Histologically-proven myocarditis has been incriminated in a single patient (W. Somerville, 1975, personal communication), and we are aware of another unpublished case.

Treatment

Certain of the aetiological factors provide important clues to the therapy of *torsade de pointes*. It is absolutely vital not to interpret the appearances as representing ventricular hyperexcitability, for quinidine and similar agents given on this assumption may disastrously aggravate the arrhythmia (Brochier *et al.*, 1972). We have on two occasions had to use direct-current countershock when *torsade de pointes* led to ventricular fibrillation (Krikler *et al.*, 1976; Evans *et al.*, 1976), but this has only a temporary effect. Clearly the underlying situation must be corrected—for example, potassium should be replaced and quinidine or other drugs withdrawn—but urgent action is needed upon diagnosis. The first step should be to infuse isoprenaline intravenously to shorten repolarization time and thus avoid a state of asynchronous depolarization; while this is being done cardiac pacing may be instituted. When heart block is the underlying cause of the arrhythmia this is the definitive therapy; when other causes are present it helps while such corrective measures as are feasible—for example, potassium infusion—are undertaken. Right atrial pacing at 100 to 120 beats a minute usually suffices, the increased rate shortening repolarization and providing less opportunity for re-entry (Brochier *et al.*, 1972); if there is AV block the ventricle must of course be paced.

Torsade de pointes can thus be seen to constitute a definable arrhythmia with important aetiological, pathogenetic, diagnostic, and therapeutic features: its recognition is not an academic matter because it requires urgent treatment quite different from that of classical ventricular tachycardia.

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